FLACCID QUADRIPLEGIA ASSOCIATED WITH HYPERPOTASSÆMIA*

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IT IS WELL KNOWN that potassium is a potentially toxic substance. Clinical effects of hyperpotassæmia have been described, but there is no pathognomonic picture. The condition has often been recognized by characteristic electrocardiographic appearances. The object of this paper is to report and discuss two cases with rapidly progressive quadriplegia associated with hyperpotassæmia.

CASE 1

P.O., male, age 21 years. Admitted 18/1/52. Three years previously he had been rejected for entry into the R.A.F. because of "bad kidneys", but had remained in apparently good health until 6 weeks before admission, when he had complained of swelling of his face, shortness of breath and diminishing urine output. He had not been given any medicine containing potassium. He was distressed and obviously dehydrated. The respirations were shallow. He was unable to move his arms or legs, but could make feeble movements of fingers and toes. All the deep reflexes were absent and the plantar responses were flexor. There were no other abnormal signs. The urine, which was alkaline, contained albumen, but no other abnormal constituents.

Laboratory investigations.—Alk. reserve 19.7 vol. %; S. sodium 316 mgm. %; S. potass. 40 mgm. %; Blood urea 480; Hb. 7.0 gm. %; P.C.V. (hæmatocrit) 19%; R.B.C. 2,530,000 per c.mm. C.S.F.: No pleocytosis; Cl. 694 mgm. %; Protein 30 mgm. %; Glucose 90 mgm. %.

E.C.G. (Fig. 1) showed defective atrioventricular and intraventricular conduction with high T waves. It was apparent that this patient was suffering from renal failure with hyperpotassæmia and several unusual features, and it was possible that the alkaline urine was a manifestation of the kidneys' inability to conserve base. The absence of hyperpnœa in severe acidosis is attributed to paresis of the respiratory muscles.

Treatment.—(1) 1/6 molar sodium lactate was given to correct acidosis. (2) 20% glucose containing 20 c.c. of 10% calcium gluconate per litre with 10 units of soluble insulin 4 hourly was administered to increase glycogen production and storage, thereby lowering the serum potassium. (3) The amount of fluid was controlled by frequent estimations of the plasma specific gravity.

Progress.—After 14 hours he was able to move all limbs, although weakness was still marked. At this time serum potassium had fallen to 28.8 mgm.% and the alkali reserve was 26 vol. %. On the third day he developed convulsions associated with hypoglycæmia: paralysis recurred (S. potass. 34 mgm. %) and he died. Autopsy (Dr. B. E. Tomlinson) showed appearances characteristic of chronic glomerulonephritis.

CASE 2

T.M., male, age 18 years. Admitted 4/4/52. Six months before admission he had complained of excessive fatigue, but examination at this time showed no abnormalities and 4 months later he was passed fit for military training. Two weeks before admission he developed a quinsy, but although this responded satisfactorily to treatment, increasing listlessness developed. Two days before admission he lost the use of his limbs for a few minutes. This recurred and persisted on the day of admission.

He was sallow, but did not appear abnormally pigmented and no pigmentation was seen in the mouth. Dehydration was marked. Systolic blood pressure was 90 mm. Hg. The diastolic pressure could not be estimated. There was complete flaccid paralysis of all limbs. The deep reflexes were absent, sensation was not impaired and the plantar reflexes were flexor. The clinical similarity between this and the previous case suggested a presumptive diagnosis of potassium intoxication. Since there was no evident renal dysfunction it was thought that he was suffering from Addison's disease with a crisis precipitated by the tonsillar infection. An immediate electrocardiograph showed appearances com-

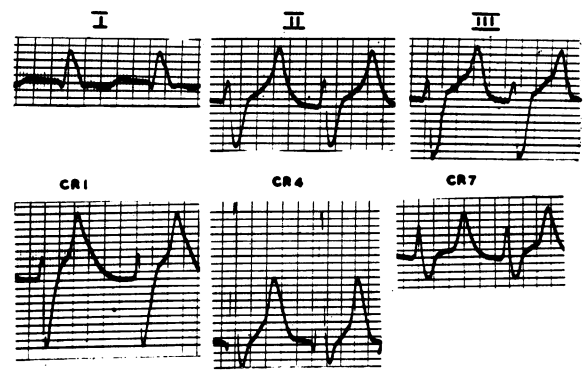

Fig. 1-A.—Serum potassium 40 mgm. %

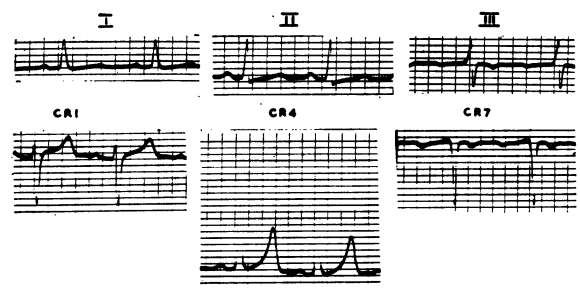

Fig. 1-B.—Serum potassium 28.8 mgm. %.

patible with hyperpotassæmia (Fig. 2). The serum potassium was 38 mgm. %. Serum sodium 245 mgm. %. Hb. 14.7 gm. %. P.C.V. (Hæmatocrit) 54%. Plasma S.G. 1.034.

Treatment.—5% glucose in physiological saline was given continuously for 3½ days (Total 6 litres).

Day 1. Cortisone 100 mgm. I.M. and Eschatin 20 c.c. 4 hourly. Day 2. Cortisine 30 mgm. I.M. and Eschatin 10 c.c. 8 hourly. Day 3. Cortisone 25 mgm. I.M. and Eschatin 8 c.c. 8 hourly.

Days 1-4 DOCA 10 mgm. b.d. Days 5-9 DOCA 10 mgm. daily. Days 10-28 DOCA 2 mgm. daily, when 4 100 mgm. pellets were implanted.

Progress.—Recovery of voluntary muscular power was rapid. At the end of three hours he was able to hold up a newspaper, and on the day after admission he was able to sit up in bed. An electrocardiograph 28 hours after admission showed no abnormalities. The biochemical changes are summarized in Table I.

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DISCUSSION

Under normal conditions abnormal retention of potassium is unlikely to occur since the kidneys can readily excrete the excess that is usually taken in the diet. In severe dehydration and sodium loss, a shift of interstitial and finally of intracellular potassium into the blood stream takes place. Potassium intoxication is therefore likely to occur if there is deficient renal function with oliguria, or in abnormal metabolic states. The administration of potassium-containing medicines in these circumstances is dangerous and should be forbidden whenever the urinary output is greatly diminished.

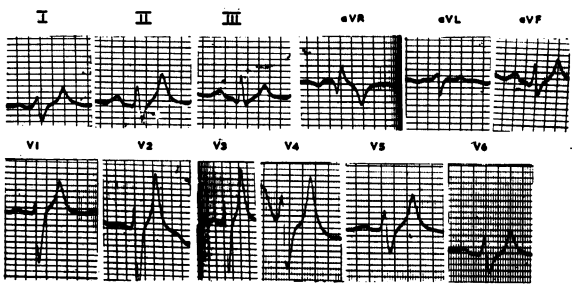

Fig. 2-A.—Serum potassium 38 mgm. %.

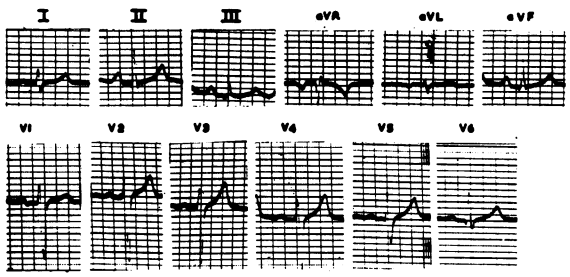

Fig. 2-B.—Serum potassium 30 mgm. %.

Keith *et al.*[1] reporting 13 cases of potassium intoxication showed that it is frequently associated with paræsthesia, weakness and shock-like states. In none of their cases did paralysis occur. Finch[2] reported 2 cases with paralysis and Merrill *et al.*[3] reported 3 more, but in all these symptoms suggestive of hyperpotassæmia developed before the onset of paralysis.

The etiology of the paralysis is not clear. In our first case complete paralysis was present with a level of 40 mgm. % and there was recovery of voluntary muscular power when the level had reached 28.8 mgm. %. But in the second case the parallel between paralysis and hyperpotassæmia was not so close. A similar lack of absolute correlation between the potassium level and the electrocardiographic changes has been noted (Merrill[3]). These workers showed that a low serum sodium enhanced the effect of a raised serum potassium, even though a lowered sodium alone produced no recognizable electrocardiographic effects. It has been shown (Fenn[4]), that an increase in serum potassium concentration around a nerve cell causes an intake of potassium and water into the cell and the axis cylinder becomes swollen. With sufficient potassium excess the threshold of electrical stimulation is raised and the excitability of the muscle thereby diminished.

Since potassium intoxication may be rapidly fatal, treatment must be started promptly. The use of substances known to be physiologically antagonistic to potassium has not met with marked success. Experimentally, calcium salts inhibit the toxic effects of potassium on the heart, but neither Finch[2] nor Merrill[3] found calcium definitely beneficial. A low serum sodium level and acidosis may accentuate the toxic effects of potassium. Finch[2] considered that hypertonic sodium chloride produced improvement in potassium intoxication, but this has not been confirmed. Sodium is likely to be helpful where there is associated sodium depletion, but its use in renal failure, unless required to correct the acid-base balance and then in deliberately measured amounts, is likely to be harmful.

TABLE I.

	Day 1		*Day 2*		*Day 3*
	5 p.m.	*11 p.m.*	*10 a.m.*	*12 noon*	*10 a.m.*
	mgm.	mgm.	mgm.	mgm.	mgm.
Potassium	38	38	36.5	30	18
Sodium	245	260	..	270	300
Alkali Res.	37	..	..	..	53

It is well known that extracellular potassium levels diminish following administration of glucose and insulin. Hypopotassæmia is often a complication of the treatment of diabetic coma with large amounts of insulin, when the potassium is probably removed in the process of glycogen formation and storage (Fenn[5]). In the treatment of potassium intoxication rapid improvement appears to follow the use of insulin and glucose, but when associated with Addison's disease specific treatment for the adrenal deficiency together with sodium chloride is probably sufficient. Moreover, the use of insulin is likely to be dangerous in this condition. Adrenal cortical extracts have been shown experimentally to protect animals against the toxic effects of potassium (Feil[6]) but there is as yet no supporting

evidence for this in clinical practice. Merrill *et al.*[3] noted that Doca failed to prevent the effects of potassium intoxication in a patient with renal failure.

SUMMARY

1. Two cases of potassium intoxication associated with paralysis have been described, and the risk of giving potassium in medicine to patients with oliguria stressed.

2. Administration of insulin and glucose is, so far, the most effective treatment for hyperpotassæmia. In certain cases (*e.g.*, Addison's disease) insulin is contra-indicated.

3. Acidosis and lowered serum sodium increase the toxic effects of potassium, and if present require correction.

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DIVERTICULITIS OF THE COLON: A SIX YEAR REVIEW

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THIS STUDY consists of a review of 50 cases of diverticulitis treated surgically during the six year period 1946-1951. The data was obtained by reviewing all cases diagnosed as diverticulosis or diverticulitis of the colon admitted to two general hospitals in Victoria, B.C.

A total of 451 case histories were reviewed. Tables I and II show the incidence of diverticulitis and diverticulosis, the average age at the time of diagnosis, and the sex incidence.

TABLE I.

	No. of cases	*Percentage*	*Average age*
Diverticulosis	214	47	65.8
Diverticulitis	186	41	67.9
Operative treatment	50	11	64.0
Total	451	100	65.0

Incidence.—No accurate estimate of diverticulosis in the general population can be made. It has been said to occur in 5 to 10% of people over 40 years of age.[1, 2] Ten to 15% of these will develop symptoms of diverticulitis.[3] Over 10% of this latter group will require surgical treatment.[4] It is generally agreed that diverticulitis is basically a medical problem and conservative therapy is indicated. In this series 21% of cases of diverticulitis were treated surgically.

The sex incidence is usually reported as being slightly higher in males than females.[5, 6, 7] The findings in this series show a considerable reversal of this ratio in that 65% of the cases were female. This figure is approximately the same for the diverticulitis and diverticulosis.

The average age of 65 years is higher than in other reported series.[5, 8] One-third of the surgically treated group were between 70 and 80 years of age. It is reasonable to expect that with the ever increasing life expectancy this disease will become more common.

Site.—The sigmoid colon is the site of the disease in about 85% of cases.[4] The descending colon is much less frequently involved. The transverse colon and the cæcum are rarely involved. In four of these cases the descending colon as well as the sigmoid was described as being the site of disease. In only one was the descending colon alone involved.

TABLE II.

	Male	*Female*	*Male to female*
Diverticulosis	87	127	1 to 1.5
Diverticulitis	58	128	1 to 2.2
Operative treatment	14	36	1 to 2.5

Two cases of perforation of diverticula of the cæcum were noted but have been excluded from this series. Smithwick[4] stated that diverticulitis occurred most frequently in the sigmoid because of the narrow lumen, stasis, and the pressure of solid fæcal material in this part of the large bowel. Mayo and Blunt[8] added to this the effect of the propulsive mechanism of this portion of the bowel and the tendency to spasm.

Symptomatology.—The signs and symptoms noted in these patients are those usually described. A history of constipation is almost uni-